ON THE EARLY DIAGNOSIS

OF SOME

ORGANIC DISEASES

OF THE

NERVOUS SYSTEM.

By E. C. SEGUIN, M.D.,
CLINICAL PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM IN THE COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK.

Reprinted from THE MEDICAL RECORD, *Feb.* 26, 1881.

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Probably no one would deny the desirability and utility of making an accurate diagnosis of disease at the earliest possible period, and one of the results of recent progress in the medical art is increased possibility in this direction. We can recognize diseases which, though existing, were unknown to practitioners of thirty or fifty years ago, and we can also determine the existence of some of these affections at a much earlier period of their evolution than we could ten or even five years ago. The sciences of semeiology and of diagnosis have unquestionably progressed greatly in the last generation, and this is more especially shown in the history of specialties, as ophthalmology, dermatology, gynecology, and neurology. I may be permitted to say that it is a duty and privilege of the specialist to inform the profession at large of the advances made in his department in diagnosis and therapeutics, in order to enable the general practitioner to apply the new knowledge, or the confirmed old knowledge, to the advantage of his patients.

It is with such a motive that I would call your attention to the possibility and desirability of an early diagnosis of two or three organic diseases of the nervous system. Probably I shall name no new symptoms, but will aim to call your attention to the really valuable symptoms of these affections, and to the significant grouping of these symptoms.

I have selected three affections which are now quite well known to us, and yet which, judging from my experience, are frequently ignored during long periods of their formative periods: I refer to poste-

rior spinal sclerosis (progressive locomotor ataxia), dementia paralytica, and cerebral tumor.

I. *Posterior spinal sclerosis, or progressive locomotor ataxia.*

While willing to admit the occasional occurrence of abnormal cases of this disease, in which ataxia appears with little premonition, yet I claim that the general practitioner at the present day should diagnosticate the disease in the clearly defined first stage, or pre-ataxic period, which may last from a few months to several years. The vast majority of cases exhibit this first stage, and its symptoms are peculiarly characteristic, if not pathognomonic. In general terms the symptoms of this first stage consist in peculiar pains, and in reduction or abolition of reflex movements in different parts of the body, and from a combination of these a diagnosis of great probability of accuracy can be made years before the patient's gait becomes disordered.

If we assume that nineteen out of twenty victims of posterior spinal sclerosis pass through this neuralgic or pre-ataxic stage, we will not be far out of the way.

The pains of posterior spinal sclerosis are almost pathognomonic, especially when described by an intelligent educated patient. They have the following characters:

a. The pains are vagrant; they occur in innumerable spots in the affected parts—so much so that patients who have long had them are unable to fully enumerate the localities in which they have suffered; or, rather, they can hardly name a region which has escaped.

b. The pains do not, as a rule, occur in the course or distribution of recognized nerve-trunks or branches; they are local pains, and this peculiarity may serve (with *a*) to distinguish between the pains of sclerosis and those of true neuralgia (sciatica, etc.).

c. The seat of pain is commonly in an area of skin varying in size from that of a pea to that of a small hand. In many cases pains are also referred to the muscles, to the vicinity of bones, and even to articulations and viscera.

d. The pains are paroxysmal in a completely irreg-

ular manner: they may occur every few moments for hours in one spot, or may be altogether wanting for weeks; or at times a single pain is the signal that the disease is not cured. It seems probable that the atmospheric disturbance which precedes a storm (areas of low barometer) causes an increase in this symptom, or even calls it forth.

e. The pains are sudden and vary in severity from the sensation caused by the penetration of a small knife-blade to what we may imagine to result from tearing through the tissues with a hook or large knife; or the sensation is like a painful electric shock. Perhaps most of the pain in such cases is in the shape of stabbing pains in an ovoid or round area of the skin (foot, thigh, arm, shin, etc.), repeated every few seconds for hours or even a day or two. The suffering is often such as to make the strongest-willed man writhe and shriek. The description of the pains, *i.e.*, their comparison with known sensations or physical conditions, varies greatly, according to the fertility of the patient's imagination and his command of language. From their suddenness and electric character the pains of posterior sclerosis are often called fulgurating or terebrating. The seat of pain usually is hyperalgesic, *i.e.*, painful to the lightest touch during the paroxysm; yet firm pressure sometimes gives relief.

Second.—Diminution of various reflexes throughout the body.

This is best observed in the iris and at the patellar tendon, though the constipation and imperfect micturition which are such frequent symptoms of the disease are phenomena of the same order.

a. The impairment of iritic reflex action ("pupillary reflex") was first intelligently studied in 1869 by Dr. Argyll Robertson, of Edinburgh. His observations have since been abundantly verified by numerous observers, and an exhaustive paper on the subject has been published by Prof. W. Erb, of Leipzig, in the *Archives of Medicine*, October, 1880. Robertson and others after him noticed that the pupil of tabetic patients did not dilate in the shadow and contract in the light, as do normal pupils; and they further ob-

served that during the effort of accommodation there occurred a normal pupillary contraction. In other words, the reflex iris movements were abolished, while its associated quasi-voluntary movements were preserved. These phenomena I have observed in almost all my patients suffering from posterior spinal sclerosis, and I am in the habit of calling the attention of students to the symptom. In two of the patients now under my care this condition is not present, but there have been cases of abnormal sclerosis, in which all the symptoms appeared in a most irregular manner.

The pupils in a suspected case of posterior-spinal sclerosis are to be tested in the following manner: the patient is placed, seated or standing, facing a brightly illuminated window, and told to keep his look fixed on some distant object, such as a house or tree. By alternately closing and opening the lids, or better, by shading the eyes with one's hand momentarily, it is easy to see if the pupils change diameter. It is of the utmost importance that the patient's intelligent assistance be secured, in order that his gaze shall remain adjusted for distance. In a given case the absence of reaction to light having been noted, we next hold up one finger or a small object within a foot of the patient's face and bid him look at it. At once the pupils contract and do so in proportion to the accommodative effort and the coincident convergence; when the patient looks at the distant object, and relatively or absolutely relaxes his accommodation, the pupils dilate again.

The finding of such a condition of the pupil—the existence of *Robertson pupils*, if you will allow the expression—is now considered of nearly as much importance for diagnosis as the occurrence of fulgurating pains.

b. Diminution and abolition of reflex action in the peripheral apparatuses is best studied at the knee.

We test the so-called patellar reflex or knee-reflex or patellar tendon-reflex in the following ways: the patient being seated, is told to cross one leg over the other in a natural manner, and to let the muscles relax; or seated, we place our left hand under the popliteal space, tell the patient not to help us, to let the

leg hang loose, or, in popular parlance, "dead," and lift the whole limb so that the foot swings a couple of inches above the floor; then we tap the skin over the whole of the region from the insertion of the quadriceps femoris to the tuberosity of the tibia, with one or two finger-tips applied as in percussion. The place whence a reflex quadriceps contraction is most apt to occur is about midway between the lower end of the patella and the tibial protuberance. The taps should be gentle at first, and if these fail, harder ones are to be tried. A third mode of procedure, which is very good indeed, is to seat the patient on a table so that his legs dangle some two or three inches beyond its edge; then we tap the patellar region as above described, without supporting the thigh with our left hand. The test may be well done through the patient's clothing, yet it is desirable, especially in doubtful cases, to tap the bare skin. Another important precaution is to secure the absolute relaxation of the patient's muscles, and to divert his attention from what you are doing. Even with all precautions it is sometimes next to impossible to secure this indispensable muscular relaxation. In the healthy subject this test develops a contraction of the quadriceps extensor femoris and causes an extension of the leg, or a sudden jerk. In a very early stage of posterior spinal sclerosis no contraction takes place.

I would also call attention to the occasional occurrence of reflex movements of the thigh produced by contraction of the iliac group of muscles during the knee-test. I have an example of this distant reflex action in a typical case of sclerosis of the posterior columns, in which the quadriceps does not contract at all.

While claiming very great diagnostic value for this negative symptom, I would not be understood as attaching pathognomonic significance to it, as we all know that there are a few seemingly healthy individuals in whom the patellar tendon-reflex is lacking, and also that there are other diseases which diminish or abolish it. Indeed, I may say that I recognize no pathognomonic symptom, and even in attempts to push diagnosis to an extreme delicacy,

would urge that reliance be placed on the grouping of symptoms, rather than on any one of the signs, however constant and important it may appear.

Physiologically analogous to this condition of loss of tendinous reflexes is the flabby state of the muscles in the affected parts. This is not due to any positive atrophy, as electrical tests show us marked departure from the normal reactions ; but to impairment of what physiologists call muscular tonus, a state of partial contraction or tension of muscles which is kept up by the inevitable and continued excitation of the cutaneous nerves by air, clothing, surrounding objects, etc., acting in a reflex way through the spinal cord. It has been recently claimed that this loss of muscular tonus was the most important factor in the production of the ataxic movements which characterize the second stage of the disease.

The vesical and rectal reflexes are diminished in posterior spinal sclerosis. Slow, irregular micturition is complained of by most patients, in the first stage and in the second. We usually micturate without using much volition, but the tabetic patient is obliged to strain and to try hard to pass water. Defecation is, like micturition, a semi-voluntary act, and in the late first stage of the disease in question constipation becomes more and more marked, and that through loss of the automatic or reflex action of the rectum and adjacent muscles.

The sexual act is, in my experience, frequently impaired and sometimes almost lost before the second stage sets in. The acts of erection and emission are usually brought about in a reflex manner by irritation of the skin and mucous membrane of the genitals. As a result of diminished spinal reflex action we have imperfect erections, and either premature emission, or, what is more common, I believe, very slow production of the orgasm, and impossibility of repetition within a reasonable time.

Some writers admit abnormally great sexual power in the early stage of tabes, but I am not sure to have met with more than one or two cases in which this seemed to be the case. In one of the patients, a female, I became convinced that her extraordinary

capacity for sexual intercourse was not in a strict sense pathological or pre-tabetic, but had been marked in one shape or another from childhood.

It seems reasonable at the present time to advance this general proposition: that in posterior spinal sclerosis the various reflex actions performed by means of those portions of the cord which are the seat of sclerosis, are diminished or lost; or, to put it in another way more useful for practice, it may be said that the limitations of loss of reflex action in different parts of the body accurately indicate the limits of sclerosis in the posterior sensory apparatus in the spinal axis.

Third.—The occurrence of paralysis of ocular muscles.

A very large proportion of tabetic patients tell of past or present diplopia, and in a certain number of cases the ocular paralysis precedes the pains and ataxia by several years. So true is this statement, that it has become an established practice with neurologists and ophthalmologists to suspect posterior spinal sclerosis in adults who present themselves with strabismus, diplopia, or ptosis. In such a case we should carefully question the patient about the occurrence of fulgurating pains, test the pupillary and tendinous reflexes. I need hardly add that another obligatory line of inquiry in such cases is with reference to symptoms of syphilis.

The same remarks apply to atrophy of the optic nerve, which is occasionally an early symptom.

I have not the time to refer to the gastric, laryngeal and rectal crises and the peculiar forms of arthritis which once in a while occur early in the disease.

It seems to me that, by a critical appreciation of the above symptoms in a patient, the diagnosis of the first or neuralgic stage of posterior spinal sclerosis is as certain as the diagnosis of any internal disease, not excepting such affections as pneumonia or valvular cardiac disease. Several autopsies are now on record, made during this first stage, and in these sections of the cord showed sclerosis of the posterior columns. I have one such observation of my own:

fulgurating pains for about thirty years, absence of patellar reflex while under observation (two years), dilatation of one pupil, no trace of ataxia. The sclerosis of the posterior columns in this patient's spinal cord is visible to the naked eye.

It is often objected that the pains of ataxia are not absolutely reliable for diagnosis. This may be true when the patient is stupid, or when the physician is not careful to ascertain the precise character of the pains.

The only two conditions in which pains somewhat resembling fulgurating pains occur, in my experience, are paralytic dementia and gout. In the former disease, slight fulgurating pains—"smaller" pains, if I may be allowed the expression—are described by the patients; but in many of these cases autopsy shows that, besides the cerebral lesions proper to the disease, the posterior columns of the cord exhibit pathological alterations; so that these cases are, after all, *quasi*-tabetic. The sharp pains of gout are short, stabbing pains in the skin of various parts of the body, compared by the patients to the prick of a needle, cold or hot. There is no tendency to repetition of the pain in one spot for hours or days; the sensations appear in various parts of the body, and are bearable. It is but right to add that this statement is based on very few observations, and requires verification.

The differential diagnosis of fulgurating pains from the pains of neuralgia, strictly speaking, is very easy. In neuralgia the pain is in the course and distribution of one or two (seldom) nerve-trunks and their branches; it may be paroxysmal, but does not assume the excessive irregularity of the tabetic pains—agony for a few hours, and freedom from pains for hours, days, or weeks. The hyperæsthesia in fulgurating pains is at the seats of pain. In neuralgia we find regular "tender points" along the nerve-trunk, or where its branches become superficial. The lightest touch causes pain in the painful districts in tabes, while the tenderness of nerves in neuralgia is usually demonstrable only by firm, localized pressure. Further, true neuralgia is seldom

bilateral, while it is the rule for fulgurating pains to appear on both sides of the median line—in both lower extremities, for example. A last important distinction is that neuralgia is relievable or curable, whereas fulgurating pains are practically incurable, and are fully relieved only by morphia-injections.

The confusion so often made between "rheumatism" and the first stage of sclerosis is even less pardonable. Of course no practitioner would mistake fulgurating pains for articular rheumatism; the error is with respect to "rheumatism," so-called, affecting muscular masses, and aponeuroses. In these affections the pains are usually dull, nearly constant, and distinctly aggravated by movements. Pressure must be firmly made upon the parts to produce pain, whereas in fulgurating pains the condition is one of cutaneous hyperalgesia under a slight touch. Again, this "rheumatic" condition is distinctly amenable to treatment (counter-irritants, etc.), whereas the pains of posterior spinal sclerosis are, in one sense, incurable.

II. The second disease of the nervous system to which I would direct your attention as the object of more exact and earlier diagnosis is *paralytic dementia*. By this term is meant the passive form of an affection which consists in peri-encephalitis, adhesion of the meninges, and various secondary degenerative changes in the brain and in the posterior columns of the spinal cord. Chronic peri-encephalitis also presents itself in an active or delirious form, which is known as general paralysis or paresis. In neither form is there a positive condition of paralysis at any time, except as a complication from the occurrence of cerebral hemorrhage or softening. Both the semeiological names, paralytic dementia and general paresis, are, strictly speaking, misnomers; yet we accept them as sufficient.

The semeiology of peri-encephalitis is complicated, and it would be beyond the scope of this essay to describe it in detail. I merely wish to call your attention to the symptoms which, in my opinion, are earliest in their appearance and significant of an incurable disease. These are tremors or fibrillary con-

tractions in various muscular groups, especially in the tongue, facial, and brachial muscles; a tremulous, thick, and vibratory speech; inequality of the pupils; dementia.

The tremor of paralytic dementia probably first makes it appearance in the facial and lingual muscles. It consists in non-rhythmical contractions of small muscles or of fasciculi of muscles, which are either present in the quiescent state of the features, or are excited by emotion or by the performance of a voluntary movement, as showing the tongue or the teeth. Sometimes innumerable fine fibrillary tremors cover the face, while in some cases the movements are coarser and irregular enough to merit the term choreic. The tongue exhibits both sets of tremors—the very fine fibrillary ones and the large choreic oscillations. There is also, though usually at a later stage, some shrivelling or atrophy of the tongue.

The hands are tremulous, usually in a fine semi-rhythmical way. This trembling is sometimes scarcely visible, but is perceptible as a delicate parchment-like fremitus on holding up the patient's extended fingers between ours. In the lower extremities the tremulousness is not apparent.

The speech is affected as a result of this tremor and as the result of a certain want of co-ordination in the muscles of articulation. Words are quickly spoken, with some syllables omitted or blurred, or with a terminal syllable left off. The articulate sounds which are produced are heard as vibratory or tremulous, and the speech seems thick. Patients semi-unconsciously avoid long or difficult words in conversation, and even seek roundabout ways of expressing their meaning by shorter words. Besides this vibratory tremulousness in articulation there is an imperfection in the pronunciation of words, long words especially. Remedy is pronounced "remdy;" constitution, "constution;" infallibility, "infalliby." The last syllable may be badly sounded or even omitted. I have known this characteristic speech to be the only well-marked symptom, and to be followed by dementia, exaltation, etc. Occasionally a patient comes to us complaining of this de-

fective articulation. I now recall two such cases, one of which died three years later in a German private asylum, with all the symptoms of general paralysis.

Just as spoken language is affected by the facial and lingual tremor, so is the handwriting altered by fibrillary contractions in the muscles which govern the movements of the fingers. A tremulous, jagged, wholly irregular handwriting results, and in some cases, where dementia is present, words or syllables are frequently omitted in composition.

The pupils in paralytic dementia are either very small or irregular, usually the latter. The reaction of the iritic muscle to the influence of light may be diminished or abolished.

I may here say, by way of parenthesis, that small and unequal pupils in a person of middle age, from twenty-five to sixty, should lead to an inquiry into the possible existence of one of three morbid states, viz.: paralytic dementia (or general paralysis), sclerosis of the posterior columns, cardiac or aortic disease (intrathoracic disease).

In my experience, the patellar tendon-reflex is often increased in paralytic dementia.

The dementia or failure of mental power is sometimes impossible to detect until after the more peripheral, physical symptoms have existed for some time. It is possible for the psychical symptoms to precede the physical; sometimes the two appear to develop simultaneously; usually, I believe, the physical symptoms already studied are apparent for months before the mind shows decay.

Dementia is evidenced by impairment of memory for recent events, by loss of the power of comparison, and consequently of judgment. Many of the automatic or quasi-automatic acts of every-day life which form a part of the patient's manner and individuality are badly performed or omitted. This leads to what is known as change of character in the subject; he becomes less neat in his attire or personal cleanliness; he loses his table-manners, handling his spoon, fork and knife awkwardly, soiling his clothing with drippings of food, etc. This impairment of judgment is probably one of the factors in the im-

morality and tendency to alcoholic indulgence which are so frequent in this disease.

Yet, in the midst of this increasing moral wreck, so visible to the immediate relatives of the patient, there may remain a degree of correctness in thought and success in every-day occupation which may impose upon strangers, and even upon a judge and jury. The things which the patient is in the habit of doing every day, and about which he has thought many years, such as professional work and business transactions, may be fairly well executed, while the tremors, pupillary irregularity, impaired articulation and handwriting, together with alteration of moral character, make the medical observer recognize a fatal, progressive disease of the brain. These cases come more frequently under the observation of general practitioners than under that of the specialist, whether asylum physician or neurologist. They are very frequently in our midst, and their early recognition may save much disgrace and impoverishment to families, though, alas! it does not pave the way for more successful therapy.

I would repeat, that a person exhibiting tremors of the facial muscles of the tongue and hand, a vibratory and slurred speech, angular or tremulous handwriting, and irregular, small pupils, should be suspected of having chronic peri-encephalitis or paralytic dementia. The addition of gradual failure of mind—dementia—makes the diagnosis certain. In case there should be superadded exalted notions, with maniacal attacks and epileptiform seizures, the case deserves the name of general paresis, and as such is the form more usually seen and studied by asylum physicians.

It has been claimed in the last few years by Fournier and others that cerebral syphilis, in the shape of arteritis, partial arachnitis and localized peri-encephalitis, might give rise to the symptoms of paralytic dementia. I am in accord with Dr. Julius Mickle and others in believing that it is often possible to distinguish the idiopathic from the syphilitic dementia. The latter is, comparatively, much more acute (or rather less chronic), in its de-

velopment; in it we do not observe the very fine muscular tremors as an early symptom; the pupillary disturbance consists usually of mydriasis of one side, with or without other signs of third-nerve palsy; the speech defect is a coarse thickness in pronunciation, rather than a vibratory, tremulous sound, which, when once heard, can never be forgotten. There are well-marked paralytic symptoms, usually hemiplegic, and decided epileptic phenomena in syphilitic cortical diseases. The dementia is seemingly more profound, causing an apparent imbecility with want of control over the sphincters. Altogether, the symptom-group is much more threatening in appearance, yet great improvement or even apparent cure may be obtained in very bad cases by the use of mercury and the heroic dosing by iodide of potassium. This therapeutic proving of a disease is of course valuable in practice, but logically it cannot be termed a diagnosis, and it is a reproach to the present state of our science that in several types of disease we should be obliged to resort to it.

III. The third organic disease of the nervous system which should, it seems to me, sometimes be recognized with positiveness much earlier than it now is, is *tumor of the brain*.

In making this statement I am perfectly aware that some cerebral tumors produce no distinct or special symptoms during life, and that others produce incongruous and apparently paradoxical symptom-groups. Some years ago, before the physiology of the brain was as well understood as it is now, we could offer no explanation of these perplexing cases which seemed to destroy our rules of diagnosis. To-day we have acquired an approximately correct knowledge of which portions of the brain (cerebrum especially), are excitable and capable of causing symptoms, and which are inexcitable, and may be the seat of extensive disease without clear indications. This I say without reference to the finer localization theories of the last five years. We know quite positively, for example, that extensive lesions may exist in the anterior and inferior portions of the frontal lobes, in the sphenoidal lobes, and in the

occipital lobes of the cerebrum, and in one-half of the cerebellum, without causing any symptom specially useful for diagnosis, such as will be considered later on. We have also learned, from Flechsig's researches, that the decussation of the motor tract just below the anterior pyramids of the medulla oblongata is variable in amount, and that in some cases there may be no crossing of fibres, or hardly any. This important law of variability in the pyramidal decussation enables us to correctly appreciate the rare cases in which a cerebral lesion produces symptoms (paralysis or spasm) on the same side of the body as itself—cases which have been so urgently pressed upon the profession by Brown-Séquard in the last ten years as proofs that our physiological laws of cerebral action and of the productions of symptoms were all wrong. These laws stand to-day, I believe, only strengthened by the exceptions which have been adduced.

All I wish to say is that tumors located in what we now term the excitable region of the cerebrum, or the motor zone, are capable of very early recognition.

The region which receives the name of motor zone is irregular in shape, and perhaps its limits are not yet well ascertained. In a general way we may say that it includes the median region of each hemisphere, in particular the posterior extremity of the third frontal convolution, the upper half of the second and first frontal, the ascending frontal and ascending parietal convolutions, the anterior gyri of the island of Reil, the paracentral lobule on the inner surface of the hemispheres, and, perhaps, a large part of the upper set of parietal convolutions. These are the motor convolutions, and embrace the so-called motor centres of Ferrier. Besides, we must include under the name of motor zone, or region, those fasciculi of white substance which connect the above-mentioned gyri with the crura cerebri, constituting the anterior half (or less) of the internal capsule as it passes between the nucleus lenticularis on the outer side and the nucleus caudatus and thalamus opticus on the inner side.

The succeeding remarks apply to tumors which involve any of this large expanse of cerebral sub-

stance, either in its external gray matter or in the fasciculi of white substance lying between the motor convolutions and the central gray bodies.

The symptoms which I think are characteristic of tumor in the motor zone of the hemispheres are:

Localized convulsions in peripheral muscles; equally localized paralysis of peripheral parts; neuro-retinitis or choked disk; localized headache. The symptoms are named in the order of their frequency and importance.

The initial convulsions of cerebral tumor are sometimes restricted to one side of the face, one hand, or even two fingers, or one leg. The spasm is usually tonico-clonic, but may be wholly clonic or jerky. In many cases this localized spasm is unaccompanied by loss of consciousness or vertigo, and it may remain localized in the part first affected during many attacks, extending over weeks and months of time. The patient feels the muscular contraction before it becomes evident, thus constituting a sort of aura. In some cases almost from the first, in nearly all cases after a while, the convulsion involves more muscles on one side of the body; it seems to ascend or descend, to use the patient's expressions, and there results a hemiplegic epileptic attack with loss of consciousness. Again, the attack may begin in a small peripheral part, involve the whole of one side of the body, and later affect the opposite side, thus constituting a full epileptic attack. The patient is able to watch the progress of the spasm for a number of seconds or minutes before losing consciousness or being thrown down, and we may take advantage of this peculiarity to instruct the patient in the use of the tourniquet or bracelet, placed on the limb just above the seat of first spasm, to cut short the attack by pressure.

This distribution of spasm, and its possible occurrence without loss of consciousness, are signs which most positively distinguish these symptomatic convulsions from the ordinary epilepsy which we constantly encounter.

As early as 1827, a French physician, Bravais, de-

scribed the hemiplegic form of epilepsy and showed its relation to gross cerebral disease; but it is to Hughlings Jackson, of London, that we owe the physiological study of these cases, and of cases of more limited epilepsy, and the first demonstration of the dependence of localized spasms upon limited lesions of the opposite cerebral hemisphere.

Indeed, in prosecuting these clinical and post-mortem studies, Hughlings Jackson laid the foundation for the vigorous hypothesis of cerebral localization, as Ferrier states in the dedication of his book on the "Functions of the Brain" to this illustrious physician. So far as my own experience goes, autopsies have invariably verified the theory of localized epilepsy which I have stated, and the journals of the last five or six years contain numerous corroborative cases. As the evidence now stands, chronic localized convulsions must be looked upon as almost positive indications of a localized lesion in the opposite cerebral motor zone, most probably a tumor.

What I have said of localized convulsions applies to localized paralysis. It, like spasm, may be limited to a small muscular group, or to one half of the body; it may begin in a part and gradually extend. In general terms paralytic phenomena follow in the wake of the convulsions at a distance of weeks or months, and have the same distribution.

Neuro-retinitis, or choked disk, is a frequent result of tumor within the cranium, but this symptom may, on the one hand, be absent with a large or even monstrous cerebral sarcoma, and on the other, it does not afford any indication of the locality of the tumor. The notion which was current a few years ago, that neuro-retinitis was pathognomonic of cerebral tumor, is wholly without foundation.

From my observations I am led to conclude that the occurrence of localized convulsions and paralysis, without choked disk, is valuable evidence of tumor, while choked disk without localized spasm and paralysis is merely a basis for suspecting tumor. The association of the two sets of symptoms makes up almost positive proof of the existence of a neoplasm. A diagnosis based on this symptom-group

is quite as secure as that of any other disease giving rise to local physical signs.

The value of headache, of localized cranial pain more strictly speaking, is also variable. By itself it is not strictly indicative of tumor, but with either the choked disks or with localized motor disturbance it becomes highly significant.

The co-existence of the three symptoms justifies a positive diagnosis of cerebral tumor.

Had I more time I should like to speak of the possibility of a still finer diagnosis in cases of tumor of motor districts of the brain. We are sometimes enabled, through recent advances in experimental physiology and pathological anatomy, to localize tumors within an inch or two of their actual situation, in the regions known as centres for speech, centres for the face, centres for the arm and hand, centres for the leg, and centres for both arm and leg. The future of neurological medicine is pregnant with discoveries in this direction, which will have very practical application.

My purpose in embracing the opportunity of addressing you was to make a sketch of the scientific and logical basis for progress in the direction of early diagnosis.

The affections whose semeiology we have studied —cerebral tumor, paralytic dementia, and posterior spinal sclerosis—are as yet incurable. Yet, if we can ever hope to apply remedies to them successfully, it will have to be done at the earliest moment when their recognition is possible by the general practitioner, who naturally has charge of the cases in their incipience.